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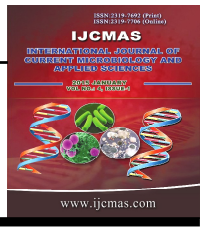

Original Research Article

Prevalence of *Helicobacter pylori* among Gastritis Patients in Sana'a, Yemen

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Keywords

H. pylori, prevalence, gastritis patients, risk factors, Sana'a, Yemen

A B S T R A C T

Helicobacter pylori is consider the most important cause of chronic gastritis and also the most important etiological factor responsible for the duodenal and gastric ulcer and has an important role in the pathogenesis of gastric cancer. This study was performed to know the prevalence of *H. pylori* and to determine some risk factors associated with infection among 56 gastritis patients attending the endoscopic department in the university of science and technology hospital, Sana'a, Yemen. The patients were 32 male and 24 female and their ages ranged from 15 to 60 years in average (37.5 years). Standard predesigned questionnaire for collection information about demographic data, history of illness, and rapid urease test (RUT) results were recorded for each patient. Our study showed an overall prevalence of *H. pylori* among gastritis patients was (35.7% n=20 of 56). Our study also revealed that there was statistical significant correlation between *H. pylori* positivity, and age of patients, social state, fat rich meal, epigastric pain, Qat chewing and antibiotic use. There was no statistical significant correlation between *H. pylori* positivity, and pepper rich meal, smoking, family history of infection and honey drinking.

Introduction

Helicobacter pylori is micro-aerophilic spiral-shaped highly motile with four to six lophotrichous flagella Gram-negative bacteria that colonize the stomach of human and cause gastrointestinal illness (Josenhans *et al.*, 2000; Fock and Ang, 2010; Nihan *et al., 2011*). *H. pylori* is contagious, although the exact route of transmission is not known (Mégraud, 1995; Cave, 1996) but Person-to-person transmission by either the oral-oral or fecal- oral route is most likely (Brown, 2000).

H. pylori is the most important etiological factor responsible for chronic gastritis, duodenal and gastric ulcer and has an important role in the pathogenesis of gastric cancer (Eugenia *et al.*, 1999). The presence of *H. pylori* induces a chronic, active inflammation in the mucosa via the release of chemokines and cytokines (such as interleukin-8, tumor necrosis factor-a, and interleukin-1b) (Veijola, 2007). This immune response is usually unable to

eradicate the organism and instead leads to persistent gastric mucosal damage (Malaty, 2007). A variety of virulence factors contribute to *H. pylori* 's ability to cause mucosal damage (Makola *et al*., 2007).

H. pylori infect at least half of the world's population and this making it the most widespread infection in the world. (Chair *et al.*, 2010). Infections are usually acquired in early childhood in all countries (Kusters *et al.*, 2006). However, the infection rate of children in developing nations is higher than in industrialized nation, probably due to poor sanitary conditions. The prevalence of *H. pylori* is higher in the developing countries and lower in the developed world, its prevalence is highly variable in relation to geography, ethnicity, age, and socioeconomic factors (Chair *et al.*, 2010).

Several techniques, both invasive (direct) and noninvasive (indirect) have been developed to diagnose *H. pylori* infection(Veijola, 2007). Rapid and correct diagnosis of *H. pylori* is critical for treatment and to prevent potential complications (Abo-Shadi *et al.*, 2013). Rapid urease test is based on the capacity of *H. pylori* if present in biopsy specimen to secrete the enzyme urease into a urea containing medium, the bacterial urease splits urea into ammonia and Co2, and pH of the medium rises and cause the color change of the pH indicator dye.

It is a quick and simple test with a sensitivity about 88-90% and a specificity close to 100% (Malfertheiner *et al*., 1996; Oona *et al*., 2004). The aims of the present study were to know the prevalence of *H. pylori* among the gastritis patients attending the endoscopic department in the university of sciences and technology hospital (USTH), Sana'a, Yemen and to determine some risk factors affecting the infection with *H. pylori*.

Material and Methods

Sample size

The sample size was calculated using the *Epi-Info*- system, a fifty specimen should be collected depend on the results of calculation but we increased the number into 56 specimen to avoid the errors. The sample size was calculated according to study carried out in Taiz, Yemen which showed prevalence of *H. pylori* infection among 80 patients was 53% (A-Ameri & Alkadasi, 2013).

Sample collection

In this cross-sectional study, we studied a total of 56 clinical biopsies collected from patients undergoing endoscopy during the period started in 8 September 2013 and ending in 22nd September 2013 at the USTH, Sana'a, Yemen.

Data collection

Standard predesigned questionnaire was used to collect information such as name, age, sex, social status, demographic data, history of illness, and tests results all above data were recorded for each patient.

Specimen collection

A total of 56 specimens (biopsies) were taken from the patients by a specialist doctor at the department of endoscopy, each biopsy was directly transfer from endoscope into the container containing urea and phenol red as an indicator. The urease produced by *H. pylori* hydrolyze urea to ammonia, which raises the pH of the medium, and changes the color of the medium, the results were read and documented.

A patient was considered to be *H. pylori* positive by RUT if the color of the specimen in the medium changed from yellow to red within 30 minutes at room temperature while considered to be *H. pylori* negative if the color of the specimen in the medium remain yellow (no change to red).

Result and Discussion

This study was performed to estimate the prevalence of *H. pylori* infection among 56 gastritis patients attending the USTH, Sana'a, Yemen. The results were analyses using SPSS version 14 and Microsoft excel 2010 software and presented in tables(1-11). A total of 56 patients 32 (57%) males and 24 (43%) female, attending the endoscopic department of USTH, Sana'a, Yemen were included in this study. The age of the patients was between 15 and 60 years with a mean age 35.5 years. A total number 20 patients gave positive result in RUT.

A patient was considered to be *H. pylori* positive by RUT if the color of the specimen in the medium changed from yellow to red within 30 minutes at room temperature while considered to be *H. pylori* negative if the color of the specimen in the medium remain yellow (no change to red). In the following we will view the results that we got it during the study support that by tables and figures for prevalence of *H. pylori* in the Sana'a city, Yemen and also the risk factors that studied with illustrating either them as significant (P value ≤ 0.05) or not significant (P value ≥ 0.05) for infected with *H. pylori* infection among gastritis patients.

Prevalence of *H. pylori* among gastritis patients in USTH

The prevalence of *H. pylori* among gastritis patients are presented in the tables (1-11). From a total of 56 specimen 20 (35.7%) were positive for *H. pylori*, while the remaining 36 (64.3%) were negative.

Prevalence of *H. pylori* among male and female

The prevalence of *H. pylori* among males and females is present in table: 1. From 56 gastritis patients 32 were male and 24 were female. The prevalence was higher in female than in male. From 24 female 12 (21.4%) had *H. pylori* while in male 8 (14.3%) of 32 had *H. pylori*.

Distribution of *H. pylori* according to patients ages

Distribution of *H. pylori* according age of patients shown in table 2. the prevalence was the highest in age group2 (21-40years) from 30 patients 16 (28%) had *H. pylori* followed by age group1 (≤20years) in which 2 (3.6%) from10 had *H. pylori* and the lowest prevalence was in age group 3 in which 2 (3.6%) from10 had *H. pylori*.

Distribution of *H. pylori* according to social status.

The prevalence of *H. pylori* according to social state shown in table 3. which showed high prevalence of *H. pylori* in married patients. from 56 patients 44 were married from which 18 (32.1%) had *H. pylori* while 12 were single from which 2 (3.2%) had *H. pylori*.

Distribution of *H. pylori* in patients with history of Pepper rich meal.

The prevalence of *H. pylori* in patients with history of Pepper rich meal which shown in table .4**.** from 28 patients who eat meal rich with pepper 12 (21.14%) had *H. pylori* while from 28 patients who do not eat meal rich with pepper 8 (14.3%) had *H. pylori* **.**

Distribution of *H. pylori* in patients with history of fat rich meal.

The prevalence of *H. pylori* in patients with history of fat rich meal which shown in table 5**.** from 10 patients who eat meal rich with fat 4 (7.1%) had *H. pylori* while from 46 patients who do not eat meal rich with fat 16 (28.6%) had *H. pylori* **.**

Epigastric pain in gastritis patients

The results of epigastric pain in gastritis patients are presented in table 6. all patients with gastritis were shown to have epigastric pain.

Distribution of *H. pylori* according to smoking.

Table 7. represent prevalence of *H. pylori* in smokers and non-smokers patients . From 56 patients 14 was smokers and 42 non-smokers. From the 14 patients who smoke 6 (10.7%) had *H. pylori.*

Distribution of *H. pylori* in Qat chewing and non-Qat chewer patients.

Table 8. represent prevalence of *H. pylori* in Qat chewer and non-Qat chewer patients. From 56 patients 34 are Qat chewers while 22 non-Qat chewers. The prevalence of *H. pylori* was higher in Qat chewers than non-Qat chewers. From 34 who chewing Qat 21(21.4%) had *H. pylori* while in non-Qat chewers 8 (14.3%) of 22 had *H. pylori.*

Prevalence of *H. pylori* and history of family infection.

The prevalence of *H. pylori* and family history of infection presented in table 9. From 56 patients 24 has family history of infection in which 12 (21.4%) had *H. pylori* while in the 32 who do not has family history of infection 8 (14.3%) had *H. pylori*.

Prevalence of *H. pylori* and antibiotics use

The prevalence *H. pylori* in the patients who use antibiotics was (32.1%) 18 of 46 patients, while in the patients who do not use antibiotics was (3.6%) 2 of 10 patients table 10.

Prevalence of *H. pylori* and honey drinking

From 56 patients, 26 were found to be honey drinkers, 10 (17.9%) of them have *H. pylori* infection, while the other 30 patients who did not drink honey, 10 (17.9%) had *H. pylori* table 11.

H. pylori is one of the most common chronic bacterial infection of humans and has a worldwide distribution. Epidemiological studies strongly suggested that more than 50% of the world's population are colonized by *H. pylori* (Mitipat *et al.*, 2005). The prevalence of *H. pylori* infection varies widely according to geographical area, patient age and socioeconomic status. Rates of infection range between 70-90% in developing countries and 25-50% in developed countries (Tanih *et al.,* 2010).

The results of our study showed an overall prevalence of *H. pylori* among gastritis patients was (35.7%, n=20 of 56). A study in Palestine (2007) agree with our results which showed that the prevalence of *H. pylori* infection according to RUT among 80 patients was 32.6% (Abu-Mugesieb, 2007). Another study from Saudi Arabia showed that the prevalence of *H. pylori* among 120 volunteer students was 35% (Almadi *et al.*, 2007). A study in Taiz, Yemen (2013) does not agree with our results which showed that the prevalence of *H. pylori* infection among 80 patients was 53%, but in their study they depended on detection of serum IgM by ELISA (A-Ameri &

Alkadasi, 2013). The variation between our finding and this study may be due to the high specificity of serum IgM test compared with biopsies RUT. Our study revealed that there is a correlation between sex and *H. pylori* positivity, the prevalence was higher in female than in male (21.4%, n=12 of 24) and (14.3%, n=8 of 32) respectively, this result is in agreement with (A-Ameri & Alkadasi, 2013; Tanih *et al.*, 2010), but disagree with other studies performed by (Abo-Shadi *et al.*, 2013; Sasidharan *et al.*, 2012).

All age groups (G) viewed correlation with *H. pylori* positivity, but the percentage differed from group to another where; G1 (3.6%, n=2 of 10), G2 (28,6%, n=16 of 30), G3 (3.6%, n=2 of 16). The high prevalence was in G2 there are many studies agree with the present study (Malik *et al.*, 2007; Abu-Ahmad *et al.*, 2011) these studies showed that the prevalence of infection increase with the age. Our study revealed that there is a correlation between a social status and *H. pylori* positivity, the prevalence in married patients was significant (32.1%, n=18 of 44) with *P. value*=(0.000). This result is agree with (Brown *et al.*, 2002).

There are studies found that the fatty acids have bactericidal effects on *H. pylori* (Sun *et al.*, 2003). But in our study there was low significant correlation between fat rich meal and *H. pylori* positivity,(7.1%, n=4 of 10), this may be due to the sample size was small to allow sufficient evaluation. In our study there is no correlation between family history of infection and *H. pylori* positivity, this is agree with (Ertem *et al.*, 2003). In our study there is a correlation between epigastric pain and *H. pylori* positivity, (35.7%, n=20 of 56). This is agree with (Javed *et al.*, 2010) who showed that epigastric pain is the most frequent symptom in gastritis patient. Our study showed that there is a correlation between Qat chewing and *H. pylori* positivity, (21.4%, n=12 of 34). This agree with (Raja'a *et al.*, 2000) who showed that this effect can be due stress that follows Qat chewing and another possible factor can be due to *H. pylori* associated with Qat chewing, beverages consumed during the session or insecticides and chemicals used for growing the Qat plants.

There are studies viewed correlation between smoking and *H. pylori* positivity, (A-Ameri & Alkadasi, 2013) but in our study there is no correlation between smoking and *H. pylori* positivity. There are studies agree with our finding (Tanih *et al.*, 2010; Abo-Shadi *et al.*, 2013) who reported that no significant difference between positive and negative *H. pylori* cases and smoking status. In our study there is correlation between antibiotic use and *H. pylori* positivity, (32.1%, n=18 of 46). There was no correlation between consuming pepper rich meal, honey drinking and *H. pylori* positivity.

Table.1 Correlation between gender and *H. pylori* infection

Sex	*H. pylori* results					
	H. pylori +ve		*H. pylori* -ve		Total	
	No.	%	No.	%	No.	%
Male	8	14.3%	24	42.9%	32	57.1%
Female	12	21.4%	12	21.4%	24	42.9%
Total	20	35.7%	36	64.3 %	56	100%

P. value=(0.000).(significant)

Table.2 Distribution of *H. pylori* according to patients ages

Age	***H. pylori* results**					
	***H. pylori* +ve**		***H. pylori* -ve**		**Total**	
	No.	%	No.	%	No.	%
G1 ≤20years	2	3.6%	8	14.3%	10	17.9%
G2(21-40)years	16	28.0%	14	25.0%	30	53.6%
G3 ≤60years	2	3.6%	14	25.0%	16	28.6%
Total	20	35.7%	36	65.3%	56	100%

≤20years: P. value=(0.111).(non-significant); (21-40)years: P. value=(0.000).(significant) ≤60years: P. value=(0.000).(significant)

Table.3 Social status and *H. pylori* infection

Social status	***H. pylori* results**					
	***H. pylori* +ve**		***H. pylori* -ve**		**Total**	
	No.	%	No.	%	No.	%
Married	18	32.1%	26	46.4%	44	78.6%
Single	2	3.6%	10	17.9%	12	21.4%
Total	20	35.7%	36	64.3%	56	100%

P. value=(0.000).(significant)

Table.4 History of consuming pepper rich meal and *H. pylori* infection

Pepper rich meal	***H. pylori* results**					
	***H. pylori* +ve**		***H. pylori* -ve**		**Total**	
	No.	%	No.	%	No.	%
Yes	12	21.4%	16	28.6%	28	50.0%
No	8	14.3%	20	35.7%	28	50.0%
Total	20	35.7%	36	64.3%	56	100%

P. value=(0.103).(non-significant)

Table.5 History of consuming fat rich meal and *H. pylori* infection

Fat rich meal	***H. pylori* results**					
	***H. pylori* +ve**		***H. pylori* -ve**		**Total**	
	No.	%	No.	%	No.	%
Yes	4	7.1%	6	10.7%	10	17.9%
No	16	28.6%	30	53.6%	46	82.1%
Total	20	35.7%	36	64.3%	56	100%

P. value=(0.032).(significant)

Table.6 Correlation between Epigastric pain and *H. pylori* infection

Epigastric pain	*H. pylori* results					
	H. pylori +ve		*H. pylori* -ve		Total	
	No.	%	No.	%	No.	%
Yes	20	35.7%	36	64.3%	56	100%
No	0	0.0%	0	0.0%	0	0.0%
Total	20	35.7%	36	64.3%	56	100%

P. value=(0.000).(significant)

Table.7 History of smoking and *H. pylori* infection

Smoking	*H. pylori* results					
	H. pylori +ve		*H. pylori* -ve		Total	
	No.	%	No.	%	No.	%
Yes	6	10.7%	8	14.3%	14	25.0%
No	14	25.0%	28	50.0%	42	75.0%
Total	20	35.7%	36	64.3%	56	100%

Table.8 History of Qat chewing and *H. pylori* infection

Qat chewing	*H. pylori* results					
	H. pylori +ve		*H. pylori* -ve		Total	
	No.	%	No.	%	No.	%
Yes	12	21.4%	22	39.3%	34	60.7%
No	8	14.3%	14	25.0%	22	39.3%
Total	20	35.7%	36	64.3%	56	100%

P. value=(0.04).(significant)

Table.9 Correlation between history of family infection and *H. pylori* infection

Family history of infection	*H. pylori* results					
	H. pylori +ve		*H. pylori* -ve		Total	
	No.	%	No.	%	No.	%
Yes	12	21.4%	12	21.4%	24	42.9%
No	8	14.3%	24	42.9%	32	57.1%
Total	20	35.7%	36	64.3%	56	100%

P. value=(0.376).(non-significant)

Table.10 History of antibiotic use and *H. pylori* infection

Antibiotic use	*H. pylori* results					
	H. pylori +ve		*H. pylori* -ve		Total	
	No.	%	No.	%	No.	%
Yes	18	32.1%	28	50.0%	46	82.1%
No	2	3.6%	8	14.3%	10	17.9%
Total	20	35.7%	36	64.3%	56	100%

P. value=(0.000).(significant)

Table.11 History of Honey drinking and *H. pylori* infection

Honey drinking	*H. pylori* results					
	H. pylori +ve		*H. pylori* -ve		Total	
	No.	%	No.	%	No.	%
Yes	10	17.9%	16	28.6%	26	46.4%
No	10	17.9%	20	35.7%	30	53.6%
Total	20	35.7%	36	64.3%	56	100%

P. value=(0.243).(non-significant)

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